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Calcitonin: Anatomy, Pathology and Therapeutic Aspects

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Introduction

Reports have questioned the physiological role of human CT in regulating calcemia since its discovery. This peptide is produced in the thyroid glands by parafollicular cells, C thyrocytes, or C cells that splice the CT/CGRP gene with other factors or hormones such as somatostatin. C cells have recently been shown to originate from the pharyngeal endoderm's ultimobranchial anlage rather than the neural crest cells as stated in all textbooks. Blood and urine CT, as well as procalcitonin, can be secreted by cells outside the thyroid glands in humans and other mammals. Taking dietary calcium intake into account, CT aids in the homeostasis of bone mineral mass during growth, lactation, and pregnancy, as well as hypo- and hypergravity [1].

Pathologic observations and experiments that led to the discovery of calcitonin, like many other hormones, were conducted on small mammals under pathologic rather than normal physiological conditions. The thyroid and parathyroid glands of dogs were removed in Sanderson's laboratory to make the first observations. Later, further CT observations included the administration of a very high dose of calcium to dogs, rats, and sheep, which resulted in a significant drop in blood calcium and regulated phosphate uptake. These findings led the authors to propose the existence of a new hormone whose critical role is to regulate the level or 'tone' of calcium in body fluids, i.e. calcemia.Originally, and incorrectly, it was thought that this hormone was released by the parathyroid glands [2].

Description

Pathologic observations and experiments that led to the discovery of calcitonin, like many other hormones, were carried out on small mammals under pathologic rather than normal physiological conditions. To make the initial observations, the thyroid and parathyroid glands of dogs were removed in Sanderson's laboratory. Later CT observations included the administration of a very high calcium dose to dogs, rats, and sheep, which resulted in a significant drop in blood calcium and regulated phosphate uptake. These findings prompted the authors to propose the existence of a new hormone whose critical role is to regulate the level or 'tone' of calcium in body fluids, i.e. calcemia. Originally, and incorrectly, it was thought that this hormone was released by the parathyroid glands [3].

Because many of these pioneering ultrastructural studies date from the 1960s, it is unfortunate that a large number of publications describing the microscopic anatomy of human and animal C cells are currently considered archives in most large libraries and biomedical citation sites. Their content is frequently available as titles on those sites, and eventually only at a high cost to today's investigators. The frustration with these unreadable archives is that important information is ignored by new investigators or considered "archaic," despite the fact that it could comfort other current data. Similar studies done in the past with ultrastructure aspects must be shamefully restarted, in animals and human samples, at the expense of people's efforts and research agency or charity grants [4].

The alpha-calcitonin gene, which encodes a small family of peptides including calcitonin, katacalcin, and calcitonin-gene related peptide, is produced by human C thyrocytes. CT and katacalcin peptides are produced from the same precursor as CGRP via alternative splicing of the CGRP/CT gene on human chromosome 11, which results in either alpha or beta CGRP isoforms, which are classified as neuropeptides. CT gene expression and activity regulation has already been studied in the unusual obese Zucker rats. The CT half-life is quite short, and the level varies with age and gender, as well as thyroid damage or excision.

CT and hormone co-adjuvants, however, are unable to prevent the progressive decay favouring osteoporosis or other pathologies due to some overall decreased actions or of their receptors and intracellular messengers. In fact, after calcidiol is transported to the proximal tubules of the kidneys and hydroxylated at the 1-a position, CT stimulates the production of calcitriol or 1, 25-dihydroxycholecalciferol. Calcidiol is converted into calcitriol, the active hormonally active metabolite of vitamin D, by the enzyme 25-hydroxyvitamin D3 1-alpha-hydroxylase. The activation of the vitamin receptor ligand raises blood calcium levels by increasing calcium uptake from the gut into the blood [5].

Conclusion

CT's function in humans is to be associated with or control the dynamics of bone growth and homeostasis during foetal, adolescent, adult, and ageing. It appears to predominate during periods of high calcium demands associated with bone remodelling, particularly after fractures, and the physiologic calcemia required during lactation. Some animal studies suggest that CT acts on the kidneys to promote increased production of the active form of vitamin D to help meet the high body calcium demands, as well as some PTH activities, by interfering with colonic reabsorption of calcium from the diet rather than using bone minerals as a mineral source.

Acknowledgement

None.

Conflict of Interest

There are no conflicts of interest by author.

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